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Components of genetic variation for resistance of strawberry to *Phytophthora cactorum* estimated using segregating seedling populations and their parent genotypes

D. V. Shaw[a]*, J. Hansen[a], G. T. Browne[b] and S. M. Shaw[a]

[a]*Plant Sciences Department, One Shields Avenue, University of California, Davis, CA, 95616; and* [b]*USDA, ARS, Crops Pathology and Genetics Research Unit, Department of Plant Pathology, University of California, Davis, CA, 95616, USA*

Strawberry (*Fragaria* × *ananassa*) seedlings from 50 bi-parental crosses among 20 elite genotypes were evaluated for resistance to *Phytophthora cactorum* after artificial inoculation. Plots of seedlings or runner plants were rated using a disease severity score and the percentage of stunted plants per plot. The distribution of cross means for percentages of plants with stunting was highly skewed; 79% of the inoculated seedlings showed some level of stunting compared to non-inoculated control seedlings, and all but one of the crosses had 50% or more stunted plants. Disease severity scores for the bi-parental crosses were normally distributed and expressed a range of variation not reflected by the percentage of visibly stunted plants. Factorial analysis based on seedling plot means demonstrated significant additive genetic variance for the disease severity score, and the additive genetic variance was 1·9 times greater than the estimated dominance variance. The cross-mean heritability was $h_c^2 = 0{\cdot}63$ for the severity score. Estimates of the additive genetic variance component using the covariance of severity scores obtained from the seedling analysis and with severity scores for their parents evaluated in a commercial environment were similar, $\sigma_a^2 = 0{\cdot}39$ and 0·30, respectively. Most of the selection response obtained through genotypic selection would thus be transferred to segregating offspring.

Keywords: *Fragaria* × *ananassa*, heritability, offspring-parent covariance, strawberry crown rot, strawberry runner plants

Introduction

Commercial strawberry cultivars (*Fragaria* × *ananassa*) exhibit a broad range of susceptibility to crown rot caused by *Phytophthora cactorum* and severe infection with this disease can be a limiting factor to successful fruit production worldwide (Maas, 1984; Browne *et al*., 2002). Several soilborne pathogens are expected to cause disease in strawberry fruiting fields established without pre-plant soil fumigation, but even with fumigation these pathogens are rarely eliminated from the soil profile at all depths (Wilhelm & Paulus, 1980; Gordon *et al*., 2002). Furthermore, *P. cactorum* is difficult if not impossible to eliminate from strawberry nursery soils or surface sources of irrigation water and can be carried into the fruit production fields on nursery stock plants with almost no symptoms. Consequently, infection by *P. cactorum* can limit productivity in most strawberry cultural systems.

*E-mail: dvshaw@ucdavis.edu

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Breeding for resistance is one strategy for managing the risk of crown and root rot caused by *P. cactorum* in strawberry. Differences in susceptibility to this pathogen among strawberry genotypes have been evaluated in several breeding populations (Bell *et al*., 1997; Eikemo *et al*., 2000; Parikka, 2003), and the range of variation observed indicates that genetic resistance can contribute to managing the consequences of this disease. Shaw *et al*. (2006) introduced a method for evaluating strawberry genotypic resistance to *P. cactorum* that mimics the path of infection expected in California production systems, i.e. infection in the propagation nursery followed by disease expression in the fruit production field after transplantation. Differences among genotypes detected using this inoculation method explained most of the variation for resistance response observed in the University of California (UC) strawberry breeding population. Furthermore, the genotypic resistance responses detected were stable over inoculation and evaluation environments; genotypic responses were also consistent over different sets of *P. cactorum* isolates (Shaw *et al*., 2006).

The demonstration that resistance to *P. cactorum* is stable across test environments and pathogen isolates is

critical to selecting resistant strawberry cultivars. However, development of efficient methods for recurrent selection of pathogen resistance over generations will also depend on the inheritance of the resistance traits and their transmission from parent to offspring (Shaw *et al*., 1996). No studies reported to date have examined the transmission of resistance in strawberry to *P. cactorum* from parent to offspring. In evaluations of strawberry seedlings for resistance to a related pathogen, *P. fragariae* (the cause of red core or red stele) seedlings were categorized as either resistant or susceptible, and the percentage of susceptible genotypes varied substantially among crosses (Mussell & Fay, 1971; Scott *et al*., 1976; Dathe, 1998). Correspondence between parental resistance class and the percentage of resistant seedlings was observed, but none of these studies attempted to quantify the inheritance of resistance to this pathogen. The distribution of genotypic means for resistance to *P. cactorum* in the California population was consistent with polygenic inheritance (Shaw *et al*., 2006), therefore the resistance expressed to this pathogen is not best evaluated using a binary classification system.

Variation in response to *P. cactorum* has been attributed to physiological differences in strawberry plant material (Eikemo *et al*., 2000). Comparison of resistance across generations in a strawberry breeding programme may be complicated by these physiological factors, because the strawberry seedlings typically used in primary genetic evaluations differ developmentally from the runner plants used in commercial production systems. The nursery inoculation system used previously to test genotypes (Shaw *et al*., 2006) was designed to simulate the infection path expected for *P. cactorum* in commercial environments, but this system was developed for runner plants and is not practically applicable to large seedling populations. Correspondence between resistance assessments derived from the nursery inoculation system and those derived from a practicable seedling inoculation system is critical for insuring the consistency of genetic effects captured due to selection response in a multi-stage selection system.

The main objective of the research reported below was to quantify the inheritance of resistance to *P. cactorum* in seedling-propagated progenies from the University of California (UC) strawberry breeding population. Specifically, genetic variance parameters were estimated that quantify the likelihood of successful transmission of resistance to this pathogen from parent genotypes to subsequent generations. A second important goal of this study was to evaluate the consistency of genetic components of variance for resistance obtained in the seedling assay with the stable genotypic variation demonstrated previously using the runner plant assay (Shaw *et al*., 2006).

Materials and methods

Plants and inoculation procedures

Strawberry seedlings from 50 bi-parental crosses and their 20 parent genotypes were evaluated for resistance to *P. cactorum* in 2005 and 2006. All of the parent genotypes were advanced selections from the UC strawberry breeding programme and the progenies evaluated for resistance were a subset of the controlled crosses generated to advance the UC breeding population in 2005. Crosses were conducted in two disconnected 5×5 factorial sets (Comstock & Robinson, 1948). All modern strawberry breeding populations originate from 54 founding genotypes and the California breeding populations trace mostly to 17 original genotypes (Sjulin & Dale, 1987). Therefore, the individuals used as parents in this experiment are necessarily related. Strawberry populations are advanced with alternating cycles of breeding and selection, and accumulated homozygosity due to mating among relatives should not affect the genetic parameter estimates obtained here (Shaw, 1995; Shaw, 1997). Furthermore, the parents in this experiment were assigned to sets, and crosses were constructed without regard for their resistance to *P. cactorum*, so the genetic variation expressed by this sample should represent the range of genetic resistance currently available in this breeding population.

Seedlings were germinated in late June 2005 and transplanted to individual cells (Jiffy #750® peat pellets) on 8 Aug 2005. On 19 Sept, 20 seedlings from each cross were treated with *P. cactorum*. For each seedling, 5 mL of V8 juice-vermiculite-oat medium (Wilcox & Mircetich, 1985) colonized by the pathogen was inserted into the cell using a 1 cm-wide spatula. The medium applied to each seedling was subdivided into two equal portions and inserted on opposite sides of each seedling's cell. In the inoculum substrate, the pathogen mainly produces mycelium and oospores (GTB, unpublished data). Four isolates of *P. cactorum* obtained from crown rot affected strawberry plants were used to produce the inoculum, and the inoculum applied to each seedling contained equal fractions of the material produced from these four isolates.

The 20 inoculated seedlings and 20 additional untreated control seedlings per cross were transplanted in a field trial near Winters, California (38°30′N, 121°59′W) on 22 Sept 2005. Two replicates, each containing 10 inoculated seedlings, were established on two-row raised beds with 1·5 m centres and 0·36 m in-row plant spacing, in an RCB design. Seedlings were established in soil previously fumigated with 2 methyl bromide: 1 trichloronitromethane (chloropicrin) at 392 kg ha^{-1} to prevent confounding of resistance to *P. cactorum* with reactions to other soil organisms. The 10 untreated seedlings per replicate from each cross were planted in an adjacent row to the infected seedlings. The untreated seedlings served as the control, and all evaluations of plant stunting or disease scores were made with reference to the adjacent control plot.

The 20 parent genotypes were evaluated using the nursery infection system described previously (Shaw *et al*., 2006), and were a subset of the genotypes used for that study. Five of the 20 genotypes were evaluated only in 2005, seven only in 2006, and eight in both years. Briefly, an infection nursery was established by planting

two cold-stored plants of each genotype in early June of the inoculation year in fumigated soil at Winters, California. V8 juice-vermiculite-oat medium permeated with *P. cactorum* was prepared as inoculum (Wilcox & Mircetich, 1985). Approximately 0·5 m^2 of soil adjacent to each nursery mother plant was cultivated and 100 mL of the medium per mother plant was incorporated into this prepared soil to a depth of 10 cm; soil inoculation dates were 27 and 26 Aug, for inoculations performed in 2004 and 2005, respectively. Runner plants that differentiated after incorporation of the *P. cactorum* medium were then set in the infested soil, and grown until fully developed.

Twelve runner plants of each parent genotype were harvested from the inoculated nursery on 16 and 14 Nov in 2004 and 2005, respectively, and transplanted at a site near Watsonville, California (lat. 36E 54′N, long. 121E 48′W) after 3 to 5 days in cold storage at 2°C. Two replicates, each containing six plants from the inoculated nursery, were established on commercial strawberry beds (two-row raised beds with 1·32 m centres and 0·36 m in-row plant spacing) in an RCB design; two runner plants for each genotype from a non-inoculated nursery at Winters were transplanted at the end of each plot on the same dates to serve as a control (Shaw *et al*., 2006).

Disease assessment

Disease symptoms were first observed on infected seedling plants in Winters on 15 Oct 2005, and on runner plants in Watsonville during late December of each evaluation year. A disease severity score was recorded for individual seedling plots at Winters on four dates and for the Watsonville runner plant trials on three or four dates in each year, beginning when symptoms were observed in the most susceptible genotypes and continuing at 3–4 week intervals until no change in symptoms was observed. The severity score was recorded on a scale of 1 to 5, where 1 = severely diseased and 5 = no symptoms of disease (i.e. comparable in appearance to non-inoculated controls). Scores 2–4 were assigned to plots with intermediate levels of symptom development: a plot score of 4 indicated mild but general stunting or moderate stunting on one or a few of the plants; plots assigned a score of 3 indicated moderate general stunting, and a score of 2 was assigned when seedlings were moderately to severely diseased, occasionally with some plant mortality. A combined severity score was obtained for each seedling or runner plant plot as the arithmetic mean of scores for all evaluation dates (Shaw *et al*., 2006); this combined score generated a continuously distributed variable that incorporates the differences among genotypes observed due to the progression of disease over time. The percentage of plants per plot with stunting symptoms or mortality was also recorded for the seedling trial, but preliminary analyses showed this variable to be strongly associated with the disease severity score ($r = -0{\cdot}82^{**}$) and values obtained for this variable were used only to provide descriptive statistics and not subjected to genetic analysis.

Genetic analyses

A factorial analysis of variance (ANOVA) was conducted for seedling disease severity scores with replications, sets, males and females in sets, and male x female interactions in sets treated as random effects. All analyses were performed using SAS procedure GLM (SAS, 1999), with expected mean squares and synthetic *F*-ratios for individual sources calculated using the RANDOM and TEST options of this procedure. The importance of all sources of variation was further resolved by obtaining estimates of variance components using the restricted maximum likelihood option (REML) of SAS procedure VARCOMP. Model components of variance were estimated using the expected mean squares in Table 1 (Searle *et al*., 1992); causal genetic components were estimated as follows (Hallauer & Miranda, 1981):

$$\sigma_a^2 = 2(\sigma_M^2 + \sigma_F^2)$$

$$\sigma_d^2 = 4(\sigma_{MF}^2)$$

In the equations above, σ_a^2 and σ_d^2 are the additive and dominance genetic variances, σ_M^2, σ_F^2, and σ_{MF}^2 are the variance components due to male parent, female parent, and their interaction within set, respectively. Estimates for σ_M^2, σ_F^2, and σ_{MF}^2 were calculated within disconnected sets and were thus pooled by the VARCOMP procedure prior to calculation of causal components.

The variance components were further used to calculate a cross-mean heritability as (Hallauer & Miranda, 1981):

$$h_c^2 = (\tfrac{1}{2}\sigma_a^2 + \tfrac{1}{4}\sigma_d^2)/[\tfrac{1}{2}\sigma_a^2 + \tfrac{1}{4}\sigma_d^2 + (\sigma_w^2)/n]$$

where σ_w^2 is the within cross variance component and is an estimate of $\tfrac{1}{2}\sigma_a^2 + \tfrac{3}{4}\sigma_d^2 + \sigma_e^2$ (Comstock & Robinson, 1948), and n is the harmonic mean of the number of plots per cross. This heritability is appropriate for predicting response to selection using bi-parental cross means. Furthermore, σ_e^2 in this expression reflects plot to plot error, and is relevant only to the experimental design used here.

An estimate of the additive genetic variance was also obtained as twice the covariance of the mean offspring and parent disease severity scores (Falconer & MacKay, 1996), or

$$\sigma_a^2 = 2\ \mathrm{COV}_{OP}$$

Table 1 Expected mean squares for ANOVA of disease severity score in strawberry seedlings inoculated with *Phytophthora cactorum*

Source	df	Expected Mean Squares
Set (S)	1	$\sigma_w^2 + 2\sigma_{F\times M}^2 + 10\sigma_F^2 + 10\sigma_M^2 + 50\sigma_R^2$
Replication (R)	1	$\sigma_w^2 + 50\sigma_R^2$
Female/set (F)	8	$\sigma_w^2 + 2\sigma_{F\times M}^2 + 10\sigma_F^2$
Male/set (M)	8	$\sigma_w^2 + 2\sigma_{F\times M}^2 + 10\sigma_M^2$
F × M/set	32	$\sigma_w^2 + 2\sigma_{F\times M}^2$
Within cross	49	σ_w^2

Because genotypic and seedling trials were conducted in separate environments, this estimate should be free of bias due to genetic by location, genetic by inoculation treatment, or genetic by plant-type interactions (Casler, 1982). The extent to which this latter estimate σ_a^2 corresponds with the parameter estimate obtained using seedling analysis indicates the relevance of seedling screens for obtaining genetic response appropriate to the development of commercial cultivar resistance and vice versa (Shaw, 1989).

Covariances were calculated directly using genotypic and bi-parental cross means. Genotypes tested at Watsonville in a single year were included after a correction for yearly mean differences estimated using a set of six cultivars present in both years (Shaw *et al*., 2006). This correction is analogous to the correction for block differences described by Schutz & Cockerham (1965), and will improve correspondence of seedling and runner plant genetic effects by eliminating variation due to differences in yearly infection rate. The mean disease severity score over 2 years was used for those parent genotypes tested in both 2005 and 2006.

Results and discussion

Little mortality was observed in the seedling trial: only 37 of the 1000 inoculated seedlings had collapsed by the end of the scoring period. The number of dead plants per bi-parental cross ranged from 0 to 6, and almost half of the total seedling mortality was concentrated in three bi-parental crosses. Conversely, plant stunting was widespread and the majority of the inoculated seedlings (79%) showed some level of stunting when compared to adjacent control plots. The distribution of bi-parental cross means for percentage stunting was highly skewed, and all but one of the crosses had 50% or more stunted plants (Fig. 1). Disease severity scores for bi-parental crosses appeared normally distributed (Fig. 2). The absence of skew in the distribution of severity scores resulted from substantial differences in the severity of plant stunting among crosses, and suggests that seedlings express a range

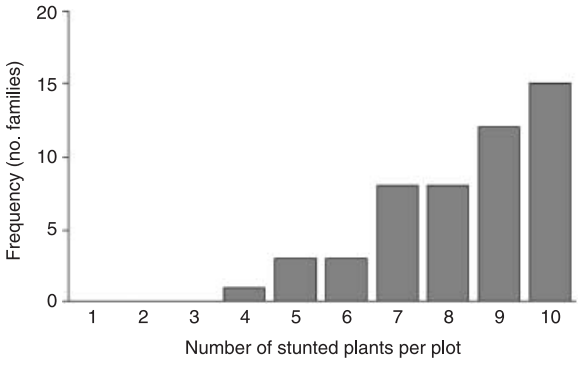

Figure 1 Distribution of bi-parental cross means for the number of seedlings inoculated with *Phytophthora cactorum* exhibiting stunting symptoms in 50 strawberry crosses evaluated in 2005.

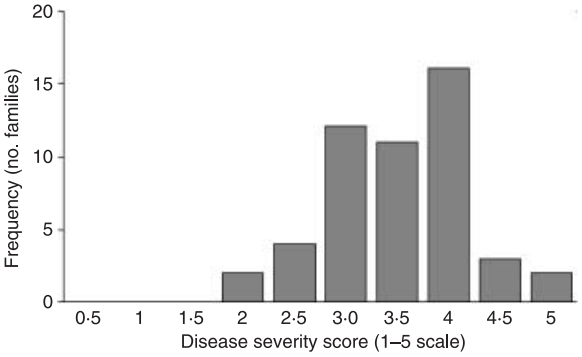

Figure 2 Distribution of bi-parental cross means for a disease severity score in 50 strawberry crosses inoculated with *Phytophthora cactorum* evaluated in 2005. The severity score was calculated as the arithmetic mean of observations collected on 3–5 dates per year, using a scale of 1 to 5, where 1 = severely diseased and 5 = no symptoms of disease (i.e. comparable in appearance to the non-inoculated controls from the same genotype).

Table 2 ANOVA results and variance component analysis for disease severity score in strawberry seedlings inoculated with *Phytophthora cactorum*

Source	df	Mean Squares
Set	1	7·18
Replication	1	0·23
Female/set (F)	8	1·32*[a]
Male/set (M)	8	1·45*
F × M/set	32	0·40
Within cross	49	0·30
σ_S^2		0·096
σ_F^2		0·092
σ_M^2		0·105
$\sigma_{F\times M}^2$		0·052
σ_w^2		0·295
σ_a^2		0·394
σ_d^2		0·208
h_c^2		0·63 (± 0·38)[b]

[a,*]indicates significance at the $P < 0{\cdot}01$ level.

[b]Standard error in parentheses calculated following Hallauer & Miranda (1981).

of genetic variation for resistance not reflected by their percentage of stunted plants. Furthermore, there was no indication of modality in either distribution that might indicate the presence of a major gene for resistance, as has been suggested for resistance of strawberry to *P. fragariae* var. *fragariae* (Van de Weg, 1997).

Highly significant ($P < 0{\cdot}01$) variances were detected for the disease severity score among both female and male parents, but significant variance was absent for any other source (Table 2). The additive genetic variance estimated for this trait was almost twice as large as the dominance variance, suggesting that most of the differences in disease sensitivity observed among parent genotypes will be transferred to their offspring. The female × male interaction

variance was not statistically significant ($P = 0{\cdot}15$), so this analysis provides no strong evidence for the presence of dominance effects at the loci affecting resistance in this experiment. The possibility of some genetic variance due to dominance indicates that genotypic evaluations may provide an imperfect assessment of parental breeding value (Falconer & MacKay, 1996).

The cross-mean heritability for disease severity score was high, $h_c^2 = 0{\cdot}63$, but substantially below its potential maximum of 1·0. The effectiveness of selection based on bi-parental cross means could be improved by increasing the number of plots per entry, for example doubling the number of 10-plant plots is expected to increase the cross-mean heritability for this population to $h_c^2 = 0{\cdot}77$. Alternatively, should future research substantiate the presence of dominance variance components for resistance to *P. cactorum* equal to half the magnitude of the additive component, a two-stage procedure of cross-mean selection followed by genotypic evaluation might be most efficient method for selection of highly resistant individual genotypes (Shaw & Gordon, 2003).

Mean disease severity scores were similar for seedling and parent evaluations (Table 3). Differing location, plant type and inoculation methods apparently had little effect on the scale of phenotypic resistance response for the two evaluation methods. The additive genetic variance for the disease severity score estimated from the covariance of parent and offspring responses was somewhat smaller than that estimated from the factorial analysis ($\sigma_a^2 = 0{\cdot}394$ and 0·300 for seedling and covariance analyses respectively, Table 3). However, these values are statistically indistinguishable, and although a direct estimate of the genetic correlation for resistance in seedling and runner plant systems is not possible from these results, the similarity of the two estimates demonstrates consistent expression of genetic resistance across the different evaluation methods. As an additional indication of consistency, the genotypic variance estimate for disease severity using a larger sample of strawberry genotypes from the UC breeding population ($\sigma_G^2 = 0{\cdot}465$, Shaw *et al.*, 2006) is consistent with an additive genetic variance of the magnitude obtained by the offspring-parent covariance procedure here and the presence of dominance variance of 50% the size of the additive genetic variance.

Both parent selection and seedling evaluation can be important components of a programme to enhance pathogen resistance in strawberry cultivars, each conducted in different stages of the breeding cycle. The *P. cactorum* screening system employed for parental genotypes here and in prior analyses is expected to simulate the path of infection realized in California production systems and should be used as the basis for evaluating the effectiveness of all other screening methods. The demonstration of high genetic correspondence between offspring-parent and offspring-only evaluation systems is critical to the utility of information derived from seedling screening systems, as is the converse in selecting parents for breeding value based on genotypic analyses. Furthermore, the genetic variation for resistance detected using prior genotypic analyses was stable over inoculation and evaluation environments, and over different sets of *P. cactorum* isolates (Shaw *et al.*, 2006). The consistency of genetic variances for seedling and runner plant resistance responses detected here suggests that genetic improvement through seedling analyses will be stable to the same factors. Taken together, these results suggest that parents selected for resistance to *P. cactorum* in genotypic screens should transmit the additive fraction of their genetic effects to seedling offspring effectively and that selection based on seedling screens should contribute to the cumulative improvement of populations leading to resistant cultivars.

Table 3 Average disease severity score and estimated additive genetic variance (± SE) following inoculations with *Phytophthora cactorum* on strawberry seedlings and their parent genotypes

	N	Severity score	σ_a^2
Seedlings[a]	50	3·32 (± 0·09)	0·394 (± 0·199)
Parent genotypes[b]	20	3·45 (± 0·16)	0·300 (± 0·146)[c]

[a]Evaluated in 2006.
[b]Evaluated in 2005 and 2006.
[c]Additive genetic variance estimates for parent genotypes were obtained from offspring-parent covariance.

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